increasingly important problem during long-term survival after heart transplantation (tx). However, the underlying mechanisms, and, in particular, the importance of extent and severity of graft rejection (R), remain undetermined. We, therefore, analysed 494 coronary angiographies (967 ± 705 days after tx, range 49 days to 9.4 years) and 5240 endomyocardial biopsies (EMB; 518 ± 648 days after tx) from 158 patients (age 46.9 ± 11.2). Patients with angiographically detectable g-CAD had significantly more episodes of R requiring augmentation of immunosuppressive therapy during the first (3.7 ± 2.7 vs. 2.2 ± 2.0, $p < 0.001$) as well as subsequent years after tx (1.2 ± 1.9 vs. 0.4 ± 0.9, $p < 0.01$) than those without evidence of g-CAD. The importance of these findings was determined by introducing known risk factors for CAD, ischemic time at tx, gender and age of donors and recipients, number of mismatches, CMV infection and drug therapy into multivariate logistic regression analysis. Data are shown as odds ratio (OR) for the development of g-CAD.

Rejection 1st year after tx (per rejection)	OR = 1.39	$p < 0.005$
Rejection ≥2 years after tx (per rejection)	OR = 1.51	$p < 0.05$
CMV infection	OR = 3.30	$p < 0.05$
Current or previous smokers	OR = 2.68	$p < 0.05$
Donor age ≥40 years	OR = 3.10	$p < 0.05$

Conclusion: R requiring augmentation of immunosuppression early and late after tx is an independent risk factor for the development of angiographically detectable g-CAD. Hence, the search for and treatment of moderate or severe R seems to be prudent even late after tx.

999-154 Obesity as a Risk Factor for Transplant Coronary Artery Disease: The IVUS Truth

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Pre-transplant obesity has been reported as an independent risk factor associated with the development of transplant coronary artery disease (TCAD); however, controversy exists. All studies have been based on angiography which has been found to be less sensitive than intravascular ultrasound (IVUS) to define TCAD. Therefore, we studied 48 cardiac transplant patients at baseline (4–6 weeks after transplant) and again at 1 year with IVUS to assess the impact of pre-transplant obesity on the development of TCAD. Obesity as previously reported is defined as body mass index greater than 27. Pre-transplant, there were 14 obese and 34 non-obese patients. There was no significant difference between the 2 groups in progression from baseline to 1 year of intimal thickness, described as maximal intimal thickness (MIT) and intimal index (II) (see table).

	Pre-Transplant Obesity Obese (14)	Non-obese (34)
ΔMIT	0.12 ± 0.07	0.18 ± 0.15
ΔII	0.05 ± 0.04	0.08 ± 0.08

One year after transplant, the entire group was redivided into obese and non-obese groups as some of the non-obese patients gained considerable weight and became obese. There were then 27 obese and 21 non-obese patients. Progression of intimal thickness was again not significantly different between these groups.

Conclusion: Pre-transplant obesity and obesity at 1 year do not appear to be associated with greater increase in intimal thickness as described by IVUS. Pre-transplant obesity does not appear to be a contraindication to transplant based on risk to develop TCAD.

1000 Electrophysiology and Hemodynamics of Atrial Fibrillation

Tuesday, March 18, 1997, Noon–2:00 p.m.
Anaheim Convention Center, Hall E
Presentation Hour: 1:00 p.m.–2:00 p.m.

1000-83 Cardiac Contractility in Atrial Fibrillation is Cycle Length Dependent

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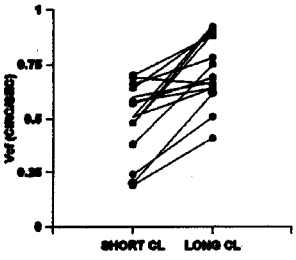
To determine the effect of cycle length (CL) on contractility in patients (pts) with atrial fibrillation (AF), we measured simultaneous beat-to-beat blood pressure, end-systolic and end-diastolic volume (EDV), circumferential velocity of fiber shortening (Vcf), and end-systolic wall stress (ESWS), in 20 (pts) with AF (mean age 71 ± 12 yrs, range 48–92 yrs; 13 M/7 F). Ejection fraction (EF) and Vcf were directly correlated with preceding CL in 90% of pts (18/20), and preceding CL was the most significant predictor of Vcf. The ratio of the preceding CL to the *pre*-preceding CL (CL1/CL2), was significantly correlated with both Vcf and EF in 85% (17/20) pts. In 75%, contractility of long CL was significantly greater than for short CL, and beats with the highest CL1/CL2 ratios also demonstrated increased contractility compared to beats with the lowest ratios. The graph demonstrates that Vcf was significantly higher at any given ESWS after longer CL (mean difference in Vcf at an ESWS of 50 g/cm² = 0.25 circ/sec, $p < 0.001$; 95% confidence intervals for the difference 0.16–0.34 circ/sec). The effect of CL on contractility was not related to EDV.



In conclusion, our data demonstrate that, for each beat in AF, variation in contractility is influenced by the duration of the preceding beat, and is independent of loading conditions. These data confirm that post-extrasystolic potentiation and mechanical restitution are important determinants of beat-to-beat variation in contractility in pts with AF.

1000-84 Left Atrial Appendage Contraction Velocities are Neither Regular in Frequency nor Uniform in Amplitude Despite Apparently Well Organized Electrical Activation in Atrial Flutter

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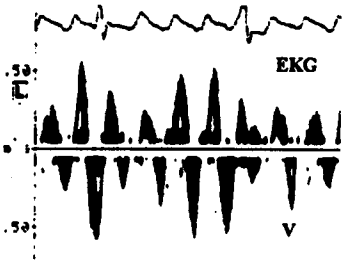
Surface and intracardiac recordings in atrial flutter demonstrate stable patterns of electrical activation with constant cycle length. Left atrial appendage (LAA) function was studied by transesophageal Doppler (V) in 11 pts with atrial flutter just prior to radiofrequency ablation. Atrial reentrant cycle length ranged from 208 to 302 ms (243 ± 30 ms) amongst the 11 pts but was *invariant* in any given pt. In contrast, LAA contraction velocity cycle length was very *irregular* resulting in a standard deviation (SD) for cycle length (measured over 10–20 cycles in any given pt) which ranged from 13 to 40 ms (5–15% of cycle length). Mean SD for LAA contraction velocity cycle length was 24 ms (10% of cycle length) for the entire group. The amplitude of LAA contraction velocities was also *nonuniform* resulting in a SD over 10–20 cycles in any given pt ranging from 4–16 cm/s (13–33% of mean contraction velocity amplitude). Mean SD for LAA contraction velocity amplitude was 9 cm/s (20% of mean contraction velocity amplitude) for the entire group. LAA filling velocities exhibited a similar degree of *nonuniformity* (mean SD for cycle length: 31 ms [13% of cycle length], mean SD for amplitude: 8 cm/s [17% of mean filling velocity] for the entire group).



Conclusions: Though atrial flutter exhibits invariant electrophysiologic periodicity, the LAA mechanical response is both irregular in cycle length and nonuniform in amplitude. Rapid *regular* atrial activation in atrial flutter may result in beat to beat *variability* of left atrial appendage mechanical function.